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=> d his

     (FILE 'HOME' ENTERED AT 14:06:24 ON 13 DEC 2006)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 14:07:00 ON 13 DEC 2006
L1            43 S "14171"
L2             4 S 14171/TI
L3             2 DUP REM L2 (2 DUPLICATES REMOVED)
L4       1527548 S KINASE?
L5             5 S L1 AND L4
L6             2 DUP REM L5 (3 DUPLICATES REMOVED)
L7        128031 S L4 AND (SERINE OR THREONINE)
L8       8068653 S CLON? OR EXPRESS? OR RECOMBINANT
L9         66403 S L7 AND L8
L10           75 S "T-P MOTIF"
L11           11 S L9 AND L10
L12            5 DUP REM L11 (6 DUPLICATES REMOVED)
L13      1018421 S (MODULAT? OR ACTIVAT? OR INHIBIT?)AND L4
L14       479153 S L8 AND L13
L15           24 S L10 AND L14
L16            7 DUP REM L15 (17 DUPLICATES REMOVED)
                 E KAPELLER-LIBERMANN
L17          114 S E2
L18            0 S L9 AND L17
L19           12 S L13 AND L17
L20            3 DUP REM L19 (9 DUPLICATES REMOVED)
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PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	AUG 09	INSPEC enhanced with 1898-1968 archive
NEWS	4	AUG 28	ADISCTI Reloaded and Enhanced
NEWS	5	AUG 30	CA(SM)/CAplus(SM) Austrian patent law changes
NEWS	6	SEP 11	CA/CAplus enhanced with more pre-1907 records
NEWS	7	SEP 21	CA/CAplus fields enhanced with simultaneous left and right truncation
NEWS	8	SEP 25	CA(SM)/CAplus(SM) display of CA Lexicon enhanced
NEWS	9	SEP 25	CAS REGISTRY(SM) no longer includes Concord 3D coordinates
NEWS	10	SEP 25	CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
NEWS	11	SEP 28	CEABA-VTB classification code fields reloaded with new classification scheme
NEWS	12	OCT 19	LOGOFF HOLD duration extended to 120 minutes
NEWS	13	OCT 19	E-mail format enhanced
NEWS	14	OCT 23	Option to turn off MARPAT highlighting enhancements available
NEWS	15	OCT 23	CAS Registry Number crossover limit increased to 300,000 in multiple databases
NEWS	16	OCT 23	The Derwent World Patents Index suite of databases on STN has been enhanced and reloaded
NEWS	17	OCT 30	CHEMLIST enhanced with new search and display field
NEWS	18	NOV 03	JAPIO enhanced with IPC 8 features and functionality
NEWS	19	NOV 10	CA/CAplus F-Term thesaurus enhanced
NEWS	20	NOV 10	STN Express with Discover! free maintenance release Version 8.01c now available
NEWS	21	NOV 13	CA/CAplus pre-1967 chemical substance index entries enhanced with preparation role
NEWS	22	NOV 20	CAS Registry Number crossover limit increased to 300,000 in additional databases
NEWS	23	NOV 20	CA/CAplus to MARPAT accession number crossover limit increased to 50,000
NEWS	24	NOV 20	CA/CAplus patent kind codes will be updated
NEWS	25	DEC 01	CAS REGISTRY updated with new ambiguity codes
NEWS	26	DEC 11	CAS REGISTRY chemical nomenclature enhanced

NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8
NEWS X25	X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that specific topic.

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FILE 'HOME' ENTERED AT 14:06:24 ON 13 DEC 2006

=> file medline embase biosis biotechds scisearch hcaplus ntis lifesci
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 14:07:00 ON 13 DEC 2006

FILE 'EMBASE' ENTERED AT 14:07:00 ON 13 DEC 2006
Copyright (c) 2006 Elsevier B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 14:07:00 ON 13 DEC 2006
Copyright (c) 2006 The Thomson Corporation

FILE 'BIOTECHDS' ENTERED AT 14:07:00 ON 13 DEC 2006
COPYRIGHT (C) 2006 THE THOMSON CORPORATION

FILE 'SCISEARCH' ENTERED AT 14:07:00 ON 13 DEC 2006
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FILE 'HCAPLUS' ENTERED AT 14:07:00 ON 13 DEC 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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FILE 'NTIS' ENTERED AT 14:07:00 ON 13 DEC 2006
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FILE 'LIFESCI' ENTERED AT 14:07:00 ON 13 DEC 2006
COPYRIGHT (C) 2006 Cambridge Scientific Abstracts (CSA)

=> s "14171"
L1 43 "14171"

=> s 14171/ti
L2 4 14171/TI

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 2 DUP REM L2 (2 DUPLICATES REMOVED)

=> d 1-2 ibib ab

L3 ANSWER 1 OF 2 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
DUPLICATE 1
ACCESSION NUMBER: 2004-12766 BIOTECHDS
TITLE: New 14171 protein kinase and nucleic acid, useful for diagnosing or treating diseases with aberrant expression of the 14171 protein kinase, such as cancer, an immunological disorder, inflammation, heart failure and hypertension;
recombinant enzyme protein production via plasmid expression in host cell for use in disease therapy

AUTHOR: KAPELLER-LIBERMANN R
PATENT ASSIGNEE: MILLENNIUM PHARM INC
PATENT INFO: US 2004048305 11 Mar 2004
APPLICATION INFO: US 2003-658904 10 Sep 2003
PRIORITY INFO: US 2003-658904 10 Sep 2003; US 2000-182096 11 Feb 2000
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2004-226195 [21]

AB DERWENT ABSTRACT:

NOVELTY - An isolated nucleic acid molecule (I) comprising a fully defined sequence of 3860 or 2355 base pairs (bp) (SEQ ID NO: 1 and 3) as given in the specification; a fragment of a fully defined sequence of 21 bp (SEQ ID NO: 21, 22 or 23) as given in the specification; or encoding a polypeptide having a fully defined sequence of 784 amino acids (SEQ ID NO: 2) as given in the specification, is new.

DETAILED DESCRIPTION - An isolated nucleic acid molecule comprises: (a) a fully defined sequence of 3860 or 2355 bp (SEQ ID NO: 1 and 3) as given in the specification; (b) a fragment of a fully defined sequence of 21 bp (SEQ ID NO: 21, 22 or 23) as given in the specification; (c) a nucleic acid molecule which encodes a polypeptide having a fully defined sequence of 784 amino acids (SEQ ID NO: 2) as given in the specification, or its fragment having at least 300 contiguous amino acids and kinase activity; or (d) the complement of (a), (b), (c), or (d). INDEPENDENT CLAIMS are also included for: (1) an expression construct comprising a recombinant nucleic acid molecule comprising the nucleic acid molecule (I); (2) a host cell comprising a recombinant nucleic acid molecule comprising the nucleic acid molecule (I); (3) an isolated polypeptide comprising: (a) a polypeptide which is encoded by a nucleic acid molecule comprising a nucleotide sequence with SEQ ID NO: 1 or 3; (b) a fragment of a polypeptide comprising the amino acid sequence of SEQ ID NO:2, where the fragment comprises at least 300 contiguous amino acids of SEQ ID NO:2 and where at least 300 contiguous amino acids have kinase activity; (c) an antigenic fragment of SEQ ID NO:2 comprising at least 15 amino acid residues of SEQ ID NO:2; or (d) a polypeptide having the amino acid sequence of SEQ ID NO:2; (4) an antibody which selectively binds to a polypeptide of (3); (5) producing a polypeptide of (3), comprising culturing the host cell of (2) under conditions in which the nucleic acid molecule is expressed; (6) a kit comprising a compound which selectively binds to a polypeptide of (3) and instructions for use; (7) a kit comprising a compound which selectively hybridizes to a nucleic acid molecule (I) and instructions for use; (8) identifying a compound which binds to a polypeptide of (3), comprising contacting a polypeptide, or a cell expressing the polypeptide with a test compound and determining whether the polypeptide binds to the test compound; (9) modulating the activity of a polypeptide of (3), comprising contacting a polypeptide or a cell expressing the polypeptide with a compound which binds to the polypeptide in a sufficient concentration to modulate the activity of the polypeptide; (10) identifying a compound which modulates the activity of a polypeptide of (3), comprising contacting the polypeptide with a test compound and determining the effect of the test compound on the activity of the polypeptide to therefore identify a compound that modulates the activity of the polypeptide; (11) identifying a subject having a disorder or at risk of developing a disorder selected from the group consisting of cancer, an immunological disorder, a viral disorder and an apoptotic disorder, comprising contacting a sample obtained from the subject comprising nucleic acid molecules with a nucleic acid probe or primer which selectively hybridizes to the nucleic acid molecule (I), and detecting in the sample the presence of a nucleic acid molecule which hybridizes to the probe or primer, therefore identifying a subject having the disorder, or at risk for developing the disorder; or comprising contacting a sample obtained from the subject comprising polypeptides with a compound which selectively binds to the polypeptide of (3), and detecting in the sample the presence of a polypeptide which binds to the compound, therefore, identifying a subject having the disorder, or at

risk for developing the disorder; and (12) treating a subject having a disorder selected from the group consisting of cancer, an immunological disorder, a viral disorder and an apoptotic disorder comprising administering to the subject an effective amount of an agent which targets the expression or activity of a nucleic acid molecule (I).

BIOTECHNOLOGY - Preferred Nucleic Acid: The nucleic acid further comprises nucleic acid sequences encoding a heterologous polypeptide. Preferred Polypeptide: The polypeptide of (3) further comprises heterologous amino acid sequences. Preferred Antibody: The antibody preferably binds to an antigenic fragment of SEQ ID NO: 2 selected from the group consisting of a fully defined sequence of 21, 20 or 21 bp (base pairs) (SEQ ID NO: 17, 18 and 19), as given in the specification. Preferred Method: The binding of the test compound to the polypeptide in the method of (8) is detected by detection of binding by direct detecting of test compound/polypeptide binding, detection of binding using a competition binding assay, or detection of binding using an assay for protein kinase-mediated phosphorylation. The activity of the polypeptide in the method of (10) is determined in a kinase assay using a 14171 kinase substrate. The nucleic acid probe or primer in the method of (11) is from a fully defined sequence of 20, 20 or 26 bp (SEQ ID NO: 9, 10 or 11) as given in the specification.

ACTIVITY - Cytostatic; Virucide; Antiinflammatory; Cardiant; Antiarrhythmic; Hypotensive. No biological data given.

MECHANISM OF ACTION - Protein-Kinase-Modulator. No biological data given.

USE - The methods and compositions of the present invention are useful for the diagnosis and/or treatment of diseases or conditions associated with aberrant expression or activity of a 14171 protein kinase, such as cancer, an immunological disorder, inflammation, heart failure, hypertension, atrial fibrillation, a viral disorder and an apoptotic disorder. They can also be used in chromosome mapping, tissue typing, predictive medicine, forensic biology and prognostic assays.

ADMINISTRATION - Dosage of the pharmaceutical composition ranges from 0.001-30 mg/kg body weight, preferably 5-6 mg/kg. Routes of administration of the pharmaceutical compositions include oral, pulmonary, intramuscular, intraperitoneal, intravenous, subcutaneous, inhalation, transdermal, nasal and rectal.

EXAMPLE - Total RNA was prepared from various human tissues by a single step extraction method using RNA STAT-60. Each RNA preparation was treated with DNase I at 37 degrees centigrade for 1 hour. DNase I treatment was determined to be complete if the sample required at least 38 PCR amplification cycles to reach a threshold level of fluorescence using beta-2 microglobulin as an internal amplicon reference. After phenol extraction cDNA was prepared from the sample using SUPERSCRIPT Choice System. A negative control of RNA without reverse transcriptase was mock reverse transcribed for each RNA sample. (62 pages)

L3 ANSWER 2 OF 2 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 2

ACCESSION NUMBER:	2003:519858 BIOSIS
DOCUMENT NUMBER:	PREV200300522904
TITLE:	14171 protein kinase, a novel human protein kinase and uses thereof.
AUTHOR(S):	Kapeller-Libermann, Rosana [Inventor, Reprint Author]
CORPORATE SOURCE:	ASSIGNEE: Millennium Pharmaceuticals, Inc.
PATENT INFORMATION:	US 6630335 20031007
SOURCE:	Official Gazette of the United States Patent and Trademark Office Patents, (Oct 7 2003) Vol. 1275, No. 1. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133 (ISSN print).
DOCUMENT TYPE:	Patent
LANGUAGE:	English
ENTRY DATE:	Entered STN: 5 Nov 2003 Last Updated on STN: 5 Nov 2003

AB The invention relates to a novel kinase nucleic acid sequence and protein. Also provided are vectors, host cells, and recombinant methods for making and using the novel molecules.

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=> d his

     (FILE 'HOME' ENTERED AT 14:06:24 ON 13 DEC 2006)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 14:07:00 ON 13 DEC 2006
L1            43 S "14171"
L2             4 S 14171/TI
L3             2 DUP REM L2 (2 DUPLICATES REMOVED)

=> s kinase?
L4      1527548 KINASE?

=> s l1 and l4
L5             5 L1 AND L4

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6             2 DUP REM L5 (3 DUPLICATES REMOVED)

=> d 1-2 ibib ab

L6    ANSWER 1 OF 2  BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
      DUPLICATE 1
ACCESSION NUMBER: 2004-12766  BIOTECHDS
TITLE:            New 14171 protein kinase and nucleic
                  acid, useful for diagnosing or treating diseases with
                  aberrant expression of the 14171 protein
                  kinase, such as cancer, an immunological disorder,
                  inflammation, heart failure and hypertension;
                     recombinant enzyme protein production via plasmid
                     expression in host cell for use in disease therapy
AUTHOR:           KAPELLER-LIBERMANN R
PATENT ASSIGNEE:  MILLENNIUM PHARM INC
PATENT INFO:      US 2004048305 11 Mar 2004
APPLICATION INFO: US 2003-658904 10 Sep 2003
PRIORITY INFO:    US 2003-658904 10 Sep 2003; US 2000-182096 11 Feb 2000
DOCUMENT TYPE:    Patent
LANGUAGE:         English
OTHER SOURCE:     WPI: 2004-226195 [21]
AB    DERWENT ABSTRACT:
      NOVELTY - An isolated nucleic acid molecule (I) comprising a fully
      defined sequence of 3860 or 2355 base pairs (bp) (SEQ ID NO: 1 and 3) as
      given in the specification; a fragment of a fully defined sequence of 21
      bp (SEQ ID NO: 21, 22 or 23) as given in the specification; or encoding a
      polypeptide having a fully defined sequence of 784 amino acids (SEQ ID
      NO: 2) as given in the specification, is new.
           DETAILED DESCRIPTION - An isolated nucleic acid molecule comprises:
      (a) a fully defined sequence of 3860 or 2355 bp (SEQ ID NO: 1 and 3) as
      given in the specification; (b) a fragment of a fully defined sequence of
      21 bp (SEQ ID NO: 21, 22 or 23) as given in the specification; (c) a
      nucleic acid molecule which encodes a polypeptide having a fully defined
      sequence of 784 amino acids (SEQ ID NO: 2) as given in the specification,
      or its fragment having at least 300 contiguous amino acids and
      kinase activity; or (d) the complement of (a), (b), (c), or (d).
      INDEPENDENT CLAIMS are also included for: (1) an expression construct
      comprising a recombinant nucleic acid molecule comprising the nucleic
      acid molecule (I); (2) a host cell comprising a recombinant nucleic acid
      molecule comprising the nucleic acid molecule (I); (3) an isolated
```

polypeptide comprising: (a) a polypeptide which is encoded by a nucleic acid molecule comprising a nucleotide sequence with SEQ ID NO: 1 or 3; (b) a fragment of a polypeptide comprising the amino acid sequence of SEQ ID NO:2, where the fragment comprises at least 300 contiguous amino acids of SEQ ID NO:2 and where at least 300 contiguous amino acids have kinase activity; (c) an antigenic fragment of SEQ ID NO:2 comprising at least 15 amino acid residues of SEQ ID NO:2; or (d) a polypeptide having the amino acid sequence of SEQ ID NO:2; (4) an antibody which selectively binds to a polypeptide of (3); (5) producing a polypeptide of (3), comprising culturing the host cell of (2) under conditions in which the nucleic acid molecule is expressed; (6) a kit comprising a compound which selectively binds to a polypeptide of (3) and instructions for use; (7) a kit comprising a compound which selectively hybridizes to a nucleic acid molecule (I) and instructions for use; (8) identifying a compound which binds to a polypeptide of (3), comprising contacting a polypeptide, or a cell expressing the polypeptide with a test compound and determining whether the polypeptide binds to the test compound; (9) modulating the activity of a polypeptide of (3), comprising contacting a polypeptide or a cell expressing the polypeptide with a compound which binds to the polypeptide in a sufficient concentration to modulate the activity of the polypeptide; (10) identifying a compound which modulates the activity of a polypeptide of (3), comprising contacting the polypeptide with a test compound and determining the effect of the test compound on the activity of the polypeptide to therefore identify a compound that modulates the activity of the polypeptide; (11) identifying a subject having a disorder or at risk of developing a disorder selected from the group consisting of cancer, an immunological disorder, a viral disorder and an apoptotic disorder, comprising contacting a sample obtained from the subject comprising nucleic acid molecules with a nucleic acid probe or primer which selectively hybridizes to the nucleic acid molecule (I), and detecting in the sample the presence of a nucleic acid molecule which hybridizes to the probe or primer, therefore identifying a subject having the disorder, or at risk for developing the disorder; or comprising contacting a sample obtained from the subject comprising polypeptides with a compound which selectively binds to the polypeptide of (3), and detecting in the sample the presence of a polypeptide which binds to the compound, therefore, identifying a subject having the disorder, or at risk for developing the disorder; and (12) treating a subject having a disorder selected from the group consisting of cancer, an immunological disorder, a viral disorder and an apoptotic disorder comprising administering to the subject an effective amount of an agent which targets the expression or activity of a nucleic acid molecule (I).

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L6 ANSWER 2 OF 2 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 2
ACCESSION NUMBER: 2003:519858 BIOSIS
DOCUMENT NUMBER: PREV200300522904
TITLE: 14171 protein kinase, a novel human protein kinase and uses thereof.
AUTHOR(S): Kapeller-Libermann, Rosana [Inventor, Reprint Author]
CORPORATE SOURCE: ASSIGNEE: Millennium Pharmaceuticals, Inc.
PATENT INFORMATION: US 6630335 20031007
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Oct 7 2003) Vol. 1275, No. 1. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133 (ISSN print).
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Nov 2003
Last Updated on STN: 5 Nov 2003
AB The invention relates to a novel kinase nucleic acid sequence and protein. Also provided are vectors, host cells, and recombinant methods for making and using the novel molecules.

=> d his

(FILE 'HOME' ENTERED AT 14:06:24 ON 13 DEC 2006)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 14:07:00 ON 13 DEC 2006
L1 43 S "14171"
L2 4 S 14171/TI
L3 2 DUP REM L2 (2 DUPLICATES REMOVED)
L4 1527548 S KINASE?
L5 5 S L1 AND L4
L6 2 DUP REM L5 (3 DUPLICATES REMOVED)

=> s l4 and (serine or threonine)
L7 128031 L4 AND (SERINE OR THREONINE)

=> s clon? or express? or recombinant
L8 8068653 CLON? OR EXPRESS? OR RECOMBINANT

```
=> s l7 and l8
L9       66403 L7 AND L8

=> s "t-p motif"
L10         75 "T-P MOTIF"

=> s l9 and l10'
MISMATCHED QUOTE 'L74''
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s l9 and l10
L11         11 L9 AND L10

=> dup rem l11
PROCESSING COMPLETED FOR L11
L12          5 DUP REM L11 (6 DUPLICATES REMOVED)

=> d -15 ibib

L12  ANSWER 1 OF 5  EMBASE  COPYRIGHT (c) 2006 Elsevier B.V. All rights
     reserved on STN                                        DUPLICATE 1
ACCESSION NUMBER:   2006271919  EMBASE
TITLE:              The low density lipoprotein receptor-related protein 6
                    interacts with glycogen synthase kinase 3 and
                    attenuates activity.
AUTHOR:             Mi K.; Dolan P.J.; Johnson G.V.W.
CORPORATE SOURCE:   G.V.W. Johnson, Dept. of Psychiatry, SC1061, University of
                    Alabama at Birmingham, 1720 7th Ave. S., Birmingham, AL
                    35294-0017, United States. gvwj@uab.edu
SOURCE:             Journal of Biological Chemistry, (24 Feb 2006) Vol. 281,
                    No. 8, pp. 4787-4794. .
                    Refs: 52
                    ISSN: 0021-9258  E-ISSN: 1083-351X  CODEN: JBCHA3
COUNTRY:            United States
DOCUMENT TYPE:      Journal; Article
FILE SEGMENT:       029     Clinical Biochemistry
                    030     Pharmacology
LANGUAGE:           English
SUMMARY LANGUAGE:   English
ENTRY DATE:         Entered STN: 1 Aug 2006
                    Last Updated on STN: 1 Aug 2006

L12  ANSWER 2 OF 5  HCAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:        2004:167474  HCAPLUS
DOCUMENT NUMBER:         140:214550
TITLE:                   Extracellular Signal-regulated Kinases 1/2
                         Are Serum-stimulated "BimEL Kinases" That
                         Bind to the BH3-only Protein BimEL Causing Its
                         Phosphorylation and Turnover
AUTHOR(S):               Ley, Rebecca; Ewings, Katherine E.; Hadfield, Kathryn;
                         Howes, Elizabeth; Balmanno, Kathryn; Cook, Simon J.
CORPORATE SOURCE:        Signalling Programme, Laboratory of Molecular
                         Signalling, The Babraham Institute, Cambridge, CB2
                         4AT, UK
SOURCE:                  Journal of Biological Chemistry (2004), 279(10),
                         8837-8847
                         CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER:               American Society for Biochemistry and Molecular
                         Biology
DOCUMENT TYPE:           Journal
LANGUAGE:                English
REFERENCE COUNT:         43    THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS
```

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:82332 HCAPLUS
DOCUMENT NUMBER: 132:204569
TITLE: ERK activation induces phosphorylation of Elk-1 at multiple S/T-P motifs to high stoichiometry
AUTHOR(S): Cruzalegui, Francisco H.; Cano, Eva; Treisman, Richard
CORPORATE SOURCE: Transcription Laboratory, Imperial Cancer Research Fund, London, WC2A 3PX, UK
SOURCE: Oncogene (1999), 18(56), 7948-7957
CODEN: ONCNES; ISSN: 0950-9232
PUBLISHER: Stockton Press
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 5 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1998377416 EMBASE
TITLE: Mitogen-activated protein kinase phosphorylates and regulates the HIV-1 Vif protein.
AUTHOR: Yang X.; Gabuzda D.
CORPORATE SOURCE: D. Gabuzda, Dana-Farber Cancer Institute, 44 Binney St., Boston, MA 02115, United States
SOURCE: Journal of Biological Chemistry, (6 Nov 1998) Vol. 273, No. 45, pp. 29879-29887. .
Refs: 60
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1998
Last Updated on STN: 10 Dec 1998

L12 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1999030926 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9811754
TITLE: Host cell-virus cross talk: phosphorylation of a hepatitis B virus envelope protein mediates intracellular signaling.
AUTHOR: Rothmann K; Schnolzer M; Radziwill G; Hildt E; Moelling K; Schaller H
CORPORATE SOURCE: Zentrum fur Molekulare Biologie Heidelberg, D-69124 Heidelberg, Germany.
SOURCE: Journal of virology, (1998 Dec) Vol. 72, No. 12, pp. 10138-47.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 15 Jan 1999
Last Updated on STN: 3 Mar 2000
Entered Medline: 30 Nov 1998

=> d 1-5 ibib ab

L12 ANSWER 1 OF 5 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 1

ACCESSION NUMBER: 2006271919 EMBASE
TITLE: The low density lipoprotein receptor-related protein 6 interacts with glycogen synthase kinase 3 and attenuates activity.
AUTHOR: Mi K.; Dolan P.J.; Johnson G.V.W.
CORPORATE SOURCE: G.V.W. Johnson, Dept. of Psychiatry, SC1061, University of Alabama at Birmingham, 1720 7th Ave. S., Birmingham, AL 35294-0017, United States. gvwj@uab.edu
SOURCE: Journal of Biological Chemistry, (24 Feb 2006) Vol. 281, No. 8, pp. 4787-4794. .
Refs: 52
ISSN: 0021-9258 E-ISSN: 1083-351X CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
030 Pharmacology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Aug 2006
Last Updated on STN: 1 Aug 2006

AB Glycogen synthase kinase 3 (GSK3) is a widely expressed Ser/Thr protein kinase that phosphorylates numerous substrates. This large number of substrates requires precise and specific regulation of GSK3 activity, which is achieved by a combination of phosphorylation, localization, and interactions with GSK3-binding proteins. Members of the Wnt canonical pathway have been shown to influence GSK3 activity. Through a yeast two-hybrid screen, we identified the Wnt canonical pathway co-receptor protein low density lipoprotein receptor-related protein 6 (LRP6) as a GSK3-binding protein. The interaction between the C terminus of LRP6 and GSK3 was also confirmed by in vitro GST pull-down assays and in situ coimmunoprecipitation assays. In vitro assays using immunoprecipitated proteins demonstrated that the C terminus of LRP6 significantly attenuated the activity of GSK3β. In situ, LRP6 significantly decreased GSK3β-mediated phosphorylation of tau at both primed and unprimed sites. Finally, it was also demonstrated that GSK3β phosphorylates the PPP(S/ T)P motifs in the C terminus of LRP6. This is the first identification of a direct interaction between LRP6 and GSK3, which results in an attenuation of GSK3 activity. .COPYRGT. 2006 by The American Society for Biochemistry and Molecular Biology, Inc.

L12 ANSWER 2 OF 5 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:167474 HCAPLUS
DOCUMENT NUMBER: 140:214550
TITLE: Extracellular Signal-regulated Kinases 1/2 Are Serum-stimulated "BimEL Kinases" That Bind to the BH3-only Protein BimEL Causing Its Phosphorylation and Turnover
AUTHOR(S): Ley, Rebecca; Ewings, Katherine E.; Hadfield, Kathryn; Howes, Elizabeth; Balmanno, Kathryn; Cook, Simon J.
CORPORATE SOURCE: Signalling Programme, Laboratory of Molecular Signalling, The Babraham Institute, Cambridge, CB2 4AT, UK
SOURCE: Journal of Biological Chemistry (2004), 279(10), 8837-8847
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Bim, a "BH3-only" protein, is expressed de novo following withdrawal of serum survival factors and promotes cell death. We have

shown previously that activation of the ERK1/2 pathway promotes phosphorylation of BimEL, targeting it for degradation via the proteasome. However, the nature of the kinase responsible for BimEL phosphorylation remained unclear. We now show that BimEL is phosphorylated on at least three sites in response to activation of the ERK1/2 pathway. By using the peptidylprolyl isomerase, Pin1, as a probe for proline-directed phosphorylation, we show that ERK1/2-dependent phosphorylation of BimEL occurs at (S/T)P motifs. ERK1/2 phosphorylates BimEL, but not BimS or BimL, in vitro, and mutation of Ser65 to alanine blocks the phosphorylation of BimEL by ERK1/2 in vitro and in vivo and prevents the degradation of the protein following activation of the ERK1/2 pathway. We also find that ERK1/2, but not JNK, can phys. associate with GST-BimEL, but not GST-BimL or GST-BimS, in vitro. ERK1/2 also binds to full-length BimEL in vivo, and we have localized a potential ERK1/2 "docking domain" lying within a 27-amino acid stretch of the BimEL protein. Our findings provide new insights into the post-translational regulation of BimEL and the role of the ERK1/2 pathway in cell survival signaling.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2000:82332 HCAPLUS

DOCUMENT NUMBER: 132:204569

TITLE: ERK activation induces phosphorylation of Elk-1 at multiple S/T-P motifs to high stoichiometry

AUTHOR(S): Cruzalegui, Francisco H.; Cano, Eva; Treisman, Richard

CORPORATE SOURCE: Transcription Laboratory, Imperial Cancer Research Fund, London, WC2A 3PX, UK

SOURCE: Oncogene (1999), 18(56), 7948-7957
CODEN: ONCNES; ISSN: 0950-9232

PUBLISHER: Stockton Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Elk-1, a member of the TCF family of Ets domain proteins, contains a C-terminal transcriptional activation domain with multiple copies of the MAPK core consensus sequence S/T-P. This region is phosphorylated by MAP kinases in vitro and in vivo, but the extent and kinetics of phosphorylation at the different sites have not been investigated in detail. We prepared antisera against the phosphorylated forms of residues T353, T363, T368, S383, S389 and T417. The antisera specifically recognize the phosphorylated Elk-1 C terminus and are specific for their cognate sites, as assessed by peptide competition and mutagenesis expts. Anal. of cells stably expressing Elk-1 in vivo shows that following serum or TPA stimulation, residues T353, T363, T368, S383, S389 and T417 become phosphorylated with similar kinetics. Mutation of any one site does not prevent phosphorylation of the others. Mutation to alanine of S383, F378 or W379, which virtually abolishes transcriptional activation by Elk-1, does not affect phosphorylation of any sites tested. Anal. of Elk-1 using two-dimensional gel electrophoresis shows that following ERK activation Elk-1 receives at least six phosphates in addition to those present prior to stimulation. We propose that the Elk-1 C-terminal regulatory domain becomes stoichiometrically phosphorylated following growth factor stimulation.

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 5 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1998377416 EMBASE

TITLE: Mitogen-activated protein kinase phosphorylates and regulates the HIV-1 Vif protein.

AUTHOR: Yang X.; Gabuzda D.

CORPORATE SOURCE: D. Gabuzda, Dana-Farber Cancer Institute, 44 Binney St., Boston, MA 02115, United States
SOURCE: Journal of Biological Chemistry, (6 Nov 1998) Vol. 273, No. 45, pp. 29879-29887. .
Refs: 60
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1998
Last Updated on STN: 10 Dec 1998

AB The human immunodeficiency virus type 1 (HIV-1) Vif protein plays a critical role in virus replication and infectivity. Here we show that Vif is phosphorylated and regulated by p44/42 mitogen-activated protein kinase (MAPK). Vif phosphorylation by MAPK was demonstrated in vitro as well as in vivo and was shown to occur on serine and threonine residues. Two-dimensional tryptic phosphopeptide mapping indicated that Vif is phosphorylated by MAPK on the same sites in vitro and in vivo. Radioactive peptide sequencing identified two phosphorylation sites, Thr96 and Ser165. These phosphorylation sites do not correspond to the known optimum consensus sequences for phosphorylation by MAPK (PX(S/T)P) nor to the minimum consensus sequence ((S/ T)P), indicating that MAPK can phosphorylate proteins at sites other than those containing the PX(S/T)P or (S/T)P motifs. Synthetic Vif peptides corresponding to the local sequences of the phosphorylation sites were not phosphorylated by MAPK, suggesting that recognition of these sites by MAPK is likely to require structural determinants outside the phosphorylation site. Mutations of the Thr96 site, which is conserved among Vif sequences from HIV-1, HIV-2, and SIV, resulted in significant loss of Vif activity and inhibition of HIV-1 replication. These results suggest that MAPK plays a direct role in regulating HIV-1 replication and infectivity by phosphorylating Vif and identify a novel mechanism for activation of HIV-1 replication by mitogens and other extracellular stimuli.

L12 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1999030926 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9811754
TITLE: Host cell-virus cross talk: phosphorylation of a hepatitis B virus envelope protein mediates intracellular signaling.
AUTHOR: Rothmann K; Schnolzer M; Radziwill G; Hildt E; Moelling K; Schaller H
CORPORATE SOURCE: Zentrum fur Molekulare Biologie Heidelberg, D-69124 Heidelberg, Germany.
SOURCE: Journal of virology, (1998 Dec) Vol. 72, No. 12, pp. 10138-47.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 15 Jan 1999
Last Updated on STN: 3 Mar 2000
Entered Medline: 30 Nov 1998

AB Phosphorylation of cytosolic pre-S domains of the duck hepatitis B virus (DHBV) large envelope protein (L) was identified as a regulatory modification involved in intracellular signaling. By using biochemical and mass spectrometric analyses of phosphopeptides obtained from metabolically radiolabeled L protein, a single phosphorylation site was identified at serine 118 as part of a PX(S/T)P

motif, which is strongly preferred by ERK-type mitogen-activated protein kinases (MAP kinases). ERK2 specifically phosphorylated L at serine 118 in vitro, and L phosphorylation was inhibited by a coexpressed MAP kinase-specific phosphatase. Furthermore, L phosphorylation and ERK activation were shown to be induced in parallel by various stimuli. Functional analysis with transfected cells showed that DHBV L possesses the ability to activate gene expression in trans and, by using mutations eliminating (S-->A) or mimicking (S-->D) serine phosphorylation, that this function correlates with L phosphorylation. These mutations had, however, no major effects on virus production in cell culture and in vivo, indicating that L phosphorylation and transactivation are not essential for hepadnavirus replication and morphogenesis. Together, these data suggest a role of the L protein in intracellular host-virus cross talk by varying the levels of pre-S phosphorylation in response to the state of the cell.

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=> s modulat? or activat? or inhibit?
<------User Break------>

=> s (modulat? or activat? or inhibit?)and l4
L13    1018421 (MODULAT? OR ACTIVAT? OR INHIBIT?) AND L4

=> d his

     (FILE 'HOME' ENTERED AT 14:06:24 ON 13 DEC 2006)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 14:07:00 ON 13 DEC 2006
L1            43 S "14171"
L2             4 S 14171/TI
L3             2 DUP REM L2 (2 DUPLICATES REMOVED)
L4       1527548 S KINASE?
L5             5 S L1 AND L4
L6             2 DUP REM L5 (3 DUPLICATES REMOVED)
L7        128031 S L4 AND (SERINE OR THREONINE)
L8       8068653 S CLON? OR EXPRESS? OR RECOMBINANT
L9         66403 S L7 AND L8
L10           75 S "T-P MOTIF"
L11           11 S L9 AND L10
L12            5 DUP REM L11 (6 DUPLICATES REMOVED)
L13      1018421 S (MODULAT? OR ACTIVAT? OR INHIBIT?)AND L4

=> s l8 and l13
L14      479153 L8 AND L13

=> s l10 and l14
L15           24 L10 AND L14

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16            7 DUP REM L15 (17 DUPLICATES REMOVED)

=> d 1-7 ibib ab

L16  ANSWER 1 OF 7  EMBASE  COPYRIGHT (c) 2006 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2006271919  EMBASE
TITLE:               The low density lipoprotein receptor-related protein 6
                     interacts with glycogen synthase kinase 3 and
                     attenuates activity.
AUTHOR:              Mi K.; Dolan P.J.; Johnson G.V.W.
CORPORATE SOURCE:    G.V.W. Johnson, Dept. of Psychiatry, SC1061, University of
                     Alabama at Birmingham, 1720 7th Ave. S., Birmingham, AL
```

35294-0017, United States. gvwj@uab.edu
SOURCE: Journal of Biological Chemistry, (24 Feb 2006) Vol. 281, No. 8, pp. 4787-4794. .
Refs: 52
ISSN: 0021-9258 E-ISSN: 1083-351X CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
030 Pharmacology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Aug 2006
Last Updated on STN: 1 Aug 2006

AB Glycogen synthase kinase 3 (GSK3) is a widely expressed Ser/Thr protein kinase that phosphorylates numerous substrates. This large number of substrates requires precise and specific regulation of GSK3 activity, which is achieved by a combination of phosphorylation, localization, and interactions with GSK3-binding proteins. Members of the Wnt canonical pathway have been shown to influence GSK3 activity. Through a yeast two-hybrid screen, we identified the Wnt canonical pathway co-receptor protein low density lipoprotein receptor-related protein 6 (LRP6) as a GSK3-binding protein. The interaction between the C terminus of LRP6 and GSK3 was also confirmed by in vitro GST pull-down assays and in situ coimmunoprecipitation assays. In vitro assays using immunoprecipitated proteins demonstrated that the C terminus of LRP6 significantly attenuated the activity of GSK3β. In situ, LRP6 significantly decreased GSK3β-mediated phosphorylation of tau at both primed and unprimed sites. Finally, it was also demonstrated that GSK3β phosphorylates the PPP(S/ T)P motifs in the C terminus of LRP6. This is the first identification of a direct interaction between LRP6 and GSK3, which results in an attenuation of GSK3 activity. .COPYRGT. 2006 by The American Society for Biochemistry and Molecular Biology, Inc.

L16 ANSWER 2 OF 7 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2004100692 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14681225
TITLE: Extracellular signal-regulated kinases 1/2 are serum-stimulated "Bim(EL) kinases" that bind to the BH3-only protein Bim(EL) causing its phosphorylation and turnover.
AUTHOR: Ley Rebecca; Ewings Katherine E; Hadfield Kathryn; Howes Elizabeth; Balmanno Kathryn; Cook Simon J
CORPORATE SOURCE: Laboratory of Molecular Signalling, Signalling Programme, The Babraham Institute, Cambridge CB2 4AT, United Kingdom.. becky.ley@bbsrc.ac.uk
SOURCE: The Journal of biological chemistry, (2004 Mar 5) Vol. 279, No. 10, pp. 8837-47. Electronic Publication: 2003-12-17. Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200407
ENTRY DATE: Entered STN: 2 Mar 2004
Last Updated on STN: 7 Jul 2004
Entered Medline: 6 Jul 2004

AB Bim, a "BH3-only" protein, is expressed de novo following withdrawal of serum survival factors and promotes cell death. We have shown previously that activation of the ERK1/2 pathway promotes phosphorylation of Bim(EL), targeting it for degradation via the proteasome. However, the nature of the kinase responsible for Bim(EL) phosphorylation remained unclear. We now show that Bim(EL) is phosphorylated on at least three sites in response to activation

of the ERK1/2 pathway. By using the peptidylprolyl isomerase, Pin1, as a probe for proline-directed phosphorylation, we show that ERK1/2-dependent phosphorylation of Bim(EL) occurs at (S/T)P motifs. ERK1/2 phosphorylates Bim(EL), but not Bim(S) or Bim(L), in vitro, and mutation of Ser(65) to alanine blocks the phosphorylation of Bim(EL) by ERK1/2 in vitro and in vivo and prevents the degradation of the protein following activation of the ERK1/2 pathway. We also find that ERK1/2, but not JNK, can physically associate with GST-Bim(EL), but not GST-Bim(L) or GST-Bim(S), in vitro. ERK1/2 also binds to full-length Bim(EL) in vivo, and we have localized a potential ERK1/2 "docking domain" lying within a 27-amino acid stretch of the Bim(EL) protein. Our findings provide new insights into the post-translational regulation of Bim(EL) and the role of the ERK1/2 pathway in cell survival signaling.

L16 ANSWER 3 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 2
ACCESSION NUMBER: 2005:89678 BIOSIS
DOCUMENT NUMBER: PREV200500087142
TITLE: Physiological role of the oxidative stress-susceptible TRPM2 Ca2+ channel in immunocytes.
AUTHOR(S): Yamamoto, Shinichiro [Reprint Author]; Shimizu, Shunichi; Ishii, Masakazu; Hagiwara, Tamio; Hara, Yuji; Negoro, Takaharu; Nishida, Motohiro; Tobe, Takashi; Mori, Yasuo; Kiuchi, Yuji
CORPORATE SOURCE: Grad Sch EngnDept Synthet Chem and BiolMol Biol Lab, Kyoto Univ, Kyoto, 6068501, Japan
SOURCE: Yakugaku Zasshi, (2004) Vol. 124, No. Suppl. 4, pp. 237-240. print.
ISSN: 0031-6903 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Mar 2005
Last Updated on STN: 2 Mar 2005

AB TRPM2 is a Ca2+ permeable channel activated by various triggers including the oxidative stress including hydrogen peroxide (H2O2). TRPM2 is expressed in immunocytes such as monocytes, lymphocytes, and neutrophils. However its physiological role is unclear. Although the activation of TRPM2 by H2O2 seems to be mediated by NAD+ and/or ADP-ribose, the activation mechanisms in the context of physiological signaling are not elucidated. Thus, We investigated the activation mechanisms of TRPM2 and the physiological role of Ca2+ influx via TRPM2 using monocytic cell line U937. Addition of H2O2 to U937 cells triggered Ca2+ influx, and the both Ca2+ influx and TRPM2 expression were attenuated by the treatment with TRPM2-specific siRNA. The H2O2-triggered TRPM2 activation was also inhibited by the treatment with ERK kinase inhibitor, PD98059. Moreover, the activation of TRPM2 recombinantly expressed in HEK293 cells was blocked by the mutation of putative phosphorylation sites (S/T-P motif) by ERK, suggesting that H2O2-triggered TRPM2 activation was controlled by ERK pathway. In U937 cells, H2O2 induced interleukin-8 (IL-8) production in extracellular Ca2+ dependent manner, which was inhibited by the treatment with TRPM2 specific siRNA and PD98059. The Ca2+ influx via TRPM2 induced by H2O2 participates in IL-8 production in U937 cells.

L16 ANSWER 4 OF 7 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2000105769 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10637505
TITLE: ERK activation induces phosphorylation of Elk-1 at multiple S/T-P motifs to high stoichiometry.
AUTHOR: Cruzalegui F H; Cano E; Treisman R

CORPORATE SOURCE: Transcription Laboratory, Imperial Cancer Research Fund, 44 Lincoln's Inn Fields, London WC2A 3PX, UK.
SOURCE: Oncogene, (1999 Dec 23) Vol. 18, No. 56, pp. 7948-57. Journal code: 8711562. ISSN: 0950-9232.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 18 Feb 2000
Last Updated on STN: 18 Feb 2000
Entered Medline: 4 Feb 2000

AB Elk-1, a member of the TCF family of Ets domain proteins, contains a C-terminal transcriptional activation domain with multiple copies of the MAPK core consensus sequence S/T-P. This region is phosphorylated by MAP kinases in vitro and in vivo, but the extent and kinetics of phosphorylation at the different sites have not been investigated in detail. We prepared antisera against the phosphorylated forms of residues T353, T363, T368, S383, S389 and T417. The antisera specifically recognize the phosphorylated Elk-1 C terminus and are specific for their cognate sites, as assessed by peptide competition and mutagenesis experiments. Analysis of cells stably expressing Elk-1 in vivo shows that following serum or TPA stimulation, residues T353, T363, T368, S383, S389 and T417 become phosphorylated with similar kinetics. Mutation of any one site does not prevent phosphorylation of the others. Mutation to alanine of S383, F378 or W379, which virtually abolishes transcriptional activation by Elk-1, does not affect phosphorylation of any sites tested. Analysis of Elk-1 using two-dimensional gel electrophoresis shows that following ERK activation Elk-1 receives at least six phosphates in addition to those present prior to stimulation. We propose that the Elk-1 C-terminal regulatory domain becomes stoichiometrically phosphorylated following growth factor stimulation.

L16 ANSWER 5 OF 7 EMBASE
ACCESSION NUMBER: 1998377416 EMBASE
TITLE: Mitogen-activated protein kinase phosphorylates and regulates the HIV-1 Vif protein.
AUTHOR: Yang X.; Gabuzda D.
CORPORATE SOURCE: D. Gabuzda, Dana-Farber Cancer Institute, 44 Binney St., Boston, MA 02115, United States
SOURCE: Journal of Biological Chemistry, (6 Nov 1998) Vol. 273, No. 45, pp. 29879-29887. .
Refs: 60
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1998
Last Updated on STN: 10 Dec 1998

AB The human immunodeficiency virus type 1 (HIV-1) Vif protein plays a critical role in virus replication and infectivity. Here we show that Vif is phosphorylated and regulated by p44/42 mitogen-activated protein kinase (MAPK). Vif phosphorylation by MAPK was demonstrated in vitro as well as in vivo and was shown to occur on serine and threonine residues. Two-dimensional tryptic phosphopeptide mapping indicated that Vif is phosphorylated by MAPK on the same sites in vitro and in vivo. Radioactive peptide sequencing identified two phosphorylation sites, Thr96 and Ser165. These phosphorylation sites do not correspond to the known optimum consensus sequences for

phosphorylation by MAPK (PX(S/T)P) nor to the minimum consensus sequence ((S/ T)P), indicating that MAPK can phosphorylate proteins at sites other than those containing the PX(S/T)P or (S/T)P motifs. Synthetic Vif peptides corresponding to the local sequences of the phosphorylation sites were not phosphorylated by MAPK, suggesting that recognition of these sites by MAPK is likely to require structural determinants outside the phosphorylation site. Mutations of the Thr96 site, which is conserved among Vif sequences from HIV-1, HIV-2, and SIV, resulted in significant loss of Vif activity and inhibition of HIV-1 replication. These results suggest that MAPK plays a direct role in regulating HIV-1 replication and infectivity by phosphorylating Vif and identify a novel mechanism for activation of HIV-1 replication by mitogens and other extracellular stimuli.

L16 ANSWER 6 OF 7 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 1999030926 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9811754
TITLE: Host cell-virus cross talk: phosphorylation of a hepatitis B virus envelope protein mediates intracellular signaling.
AUTHOR: Rothmann K; Schnolzer M; Radziwill G; Hildt E; Moelling K; Schaller H
CORPORATE SOURCE: Zentrum fur Molekulare Biologie Heidelberg, D-69124 Heidelberg, Germany.
SOURCE: Journal of virology, (1998 Dec) Vol. 72, No. 12, pp. 10138-47.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 15 Jan 1999
Last Updated on STN: 3 Mar 2000
Entered Medline: 30 Nov 1998

AB Phosphorylation of cytosolic pre-S domains of the duck hepatitis B virus (DHBV) large envelope protein (L) was identified as a regulatory modification involved in intracellular signaling. By using biochemical and mass spectrometric analyses of phosphopeptides obtained from metabolically radiolabeled L protein, a single phosphorylation site was identified at serine 118 as part of a PX(S/T)P motif, which is strongly preferred by ERK-type mitogen-activated protein kinases (MAP kinases). ERK2 specifically phosphorylated L at serine 118 in vitro, and L phosphorylation was inhibited by a coexpressed MAP kinase-specific phosphatase. Furthermore, L phosphorylation and ERK activation were shown to be induced in parallel by various stimuli. Functional analysis with transfected cells showed that DHBV L possesses the ability to activate gene expression in trans and, by using mutations eliminating (S-->A) or mimicking (S-->D) serine phosphorylation, that this function correlates with L phosphorylation. These mutations had, however, no major effects on virus production in cell culture and in vivo, indicating that L phosphorylation and transactivation are not essential for hepadnavirus replication and morphogenesis. Together, these data suggest a role of the L protein in intracellular host-virus cross talk by varying the levels of pre-S phosphorylation in response to the state of the cell.

L16 ANSWER 7 OF 7 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 5
ACCESSION NUMBER: 1995:391391 SCISEARCH
THE GENUINE ARTICLE: RC664
TITLE: COMPARATIVE-ANALYSIS OF THE TERNARY COMPLEX FACTORS ELK-1, SAP-1A AND SAP-2 (ERP/NET)
AUTHOR: PRICE M A (Reprint); ROGERS A E; TREISMAN R

CORPORATE SOURCE: IMPERIAL CANC RES FUND, TRANSCRIPT LAB, 44 LINCOLNS INN FIELDS, LONDON WC2A 3PX, ENGLAND (Reprint)
COUNTRY OF AUTHOR: ENGLAND
SOURCE: EMBO JOURNAL, (1 JUN 1995) Vol. 14, No. 11, pp. 2589-2601. ISSN: 0261-4189.
PUBLISHER: OXFORD UNIV PRESS UNITED KINGDOM, WALTON ST JOURNALS DEPT, OXFORD, ENGLAND OX2 6DP.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 45
ENTRY DATE: Entered STN: 1995
Last Updated on STN: 1995
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A transcription factor ternary complex composed of Serum Response Factor (SRF) and Ternary Complex Factor (TCF) mediates the response of the c-fos Serum Response Element (SRE) to growth factors and mitogens. Three Ets domain proteins, Elk-1, SAP-1 and ERP/NET, have been reported to have the properties of TCF. Here we compare Elk-1 and SAP-1a with the human ERP/NET homologue SAP-2. All three TCF RNAs are ubiquitously expressed at similar relative levels. All three proteins contain conserved regions that interact with SRF and the c-fos SRE with comparable efficiency, but in vitro complex formation by SAP-2 is strongly inhibited by its C-terminal sequences. Similarly, only Elk-1 and SAP-1a efficiently bind the c-fos SRE in vivo; ternary complex formation by SAP-2 is weak and is substantially unaffected by serum stimulation or v-ras co-expression. All three TCFs contain C-terminal transcriptional activation domains that are phosphorylated following growth factor stimulation. Activation requires conserved S/T-P motifs found in all the TCF family members, Each TCF activation domain can be phosphorylated in vitro by partially purifed ERK2, and ERK activation in vivo is sufficient to potentiate transcriptional activation.

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E2         114     KAPELLER/BI
E3           0 --> KAPELLER-LIBERMANN/BI
E4           1     KAPELLERADLER/BI
E5           1     KAPELLEVICH/BI
E6           3     KAPELLI/BI
E7           1     KAPELLIMESTARI/BI
E8           1     KAPELLOU/BI
E9           1     KAPELLSKAR/BI
E10          2     KAPELLY/BI
E11          2     KAPELNA/BI
E12          1     KAPELNER/BI

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L17        114 KAPELLER/BI

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     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 14:07:00 ON 13 DEC 2006
L1           43 S "14171"
L2            4 S 14171/TI
L3            2 DUP REM L2 (2 DUPLICATES REMOVED)
L4      1527548 S KINASE?
L5            5 S L1 AND L4
L6            2 DUP REM L5 (3 DUPLICATES REMOVED)
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L7       128031 S L4 AND (SERINE OR THREONINE)
L8      8068653 S CLON? OR EXPRESS? OR RECOMBINANT
L9        66403 S L7 AND L8
L10          75 S "T-P MOTIF"
L11          11 S L9 AND L10
L12           5 DUP REM L11 (6 DUPLICATES REMOVED)
L13     1018421 S (MODULAT? OR ACTIVAT? OR INHIBIT?)AND L4
L14      479153 S L8 AND L13
L15          24 S L10 AND L14
L16           7 DUP REM L15 (17 DUPLICATES REMOVED)
                E KAPELLER-LIBERMANN
L17         114 S E2

=> s l9 and l17
L18           0 L9 AND L17

=> s l13 and l17
L19          12 L13 AND L17

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PROCESSING COMPLETED FOR L19
L20           3 DUP REM L19 (9 DUPLICATES REMOVED)

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L20  ANSWER 1 OF 3     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   92147704     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 1310686
TITLE:              Phosphatidylinositol-3-kinase in isolated rat
                    adipocytes. Activation by insulin and subcellular
                    distribution.
AUTHOR:             Kelly K L; Ruderman N B; Chen K S
CORPORATE SOURCE:   Boston University Medical Center, Division of Diabetes and
                    Metabolism, Massachusetts 02118-2393.
CONTRACT NUMBER:    DK42621 (NIDDK)
SOURCE:             The Journal of biological chemistry, (1992 Feb 15) Vol.
                    267, No. 5, pp. 3423-8.
                    Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        199203
ENTRY DATE:         Entered STN: 5 Apr 1992
                    Last Updated on STN: 6 Feb 1998
                    Entered Medline: 17 Mar 1992
AB   Insulin increases phosphatidylinositol-3-kinase (PI-3-
     kinase) activity in Chinese hamster ovary cells transfected with
     human insulin receptor (Ruderman, N. B., Kapeller, R., White,
     M. F., and Cantley, L. C. (1990) Proc. Natl. Acad. Sci. U.S.A. 87,
     1411-1415). The subcellular distribution of PI-3-kinase has not
     been investigated, and it is unclear if insulin has a stimulatory effect
     on PI-3-kinase in a nonproliferating target tissue, and, if so,
     whether this effect is subject to counter-regulation. To address these
     questions, we studied the effect of insulin on PI-3-kinase
     activity in isolated rat adipocytes. Activity was measured in plasma
     membranes, intracellular membranes, and cytosol of control and
     insulin-treated adipocytes, and in anti-Tyr(P) immunoprecipitates prepared
     from these fractions and from whole cell lysates. Treatment of adipocytes
     with insulin (200 nM) caused a half-maximal increase in
     anti-Tyr(P)-immunoprecipitable PI-3-kinase activity in whole
     cell lysates within 2 min. This effect was concentration-dependent, and
     it was sensitive to inhibition by norepinephrine. In
     insulin-stimulated cells, 75% of anti-Tyr(P)-immunoprecipitable PI-3-
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kinase activity was found in the low density microsomes. This fraction also exhibited the highest specific activity of PI-3-kinase, and insulin caused a further increase in this activity. Anti-Tyr(P)-immunoprecipitable PI-3-kinase activity was also found in the plasma membranes of insulin-treated cells, but this accounted for only a minor portion of the total and anti-Tyr(P)-immunoprecipitable PI-3-kinase activity. The majority of PI-3-kinase activity (90%) in control cells was cytosolic, but this was not increased in response to insulin nor was it anti-Tyr(P)-immunoprecipitable. These data demonstrate that insulin increases the activity of PI-3-kinase in adipocytes and this effect is subject to inhibition by a physiological antagonist of insulin action. The data also indicate that the effect of insulin to increase PI-3-kinase activity is expressed primarily in the low density intracellular membranes and to a lesser extent in the plasma membranes.

L20 ANSWER 2 OF 3 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 92112787 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1309768
TITLE: Insulin stimulation of phosphatidylinositol 3-kinase activity maps to insulin receptor regions required for endogenous substrate phosphorylation.
AUTHOR: Backer J M; Schroeder G G; Kahn C R; Myers M G Jr; Wilden P A; Cahill D A; White M F
CORPORATE SOURCE: Joslin Diabetes Center, Department of Medicine, Brigham and Women's Hospital, Boston, Massachusetts 02215.
CONTRACT NUMBER: DK33201 (NIDDK)
DK36836 (NIDDK)
DK38712 (NIDDK)
SOURCE: The Journal of biological chemistry, (1992 Jan 15) Vol. 267, No. 2, pp. 1367-74.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 8 Mar 1992
Last Updated on STN: 3 Mar 2000
Entered Medline: 14 Feb 1992

AB We have studied the phosphatidylinositol 3-kinase (PtdIns 3-kinase) in insulin-stimulated Chinese hamster ovary (CHO) cells expressing normal (CHO/IR) and mutant human insulin receptors. Insulin stimulation of CHO/IR cells results in an increase in PtdIns 3-kinase activity associated with anti-phosphotyrosine (alpha PY) immunoprecipitates, which has been previously shown to correlate with the in vivo production of PtdIns(3,4)P2, and PtdIns(3,4,5)P3 (Ruderman, N., Kapeller, R., White, M.F., and Cantley, L.C. (1990) Proc. Natl. Acad. Sci. U.S.A. 87, 1411-1415). Stimulation was maximal within 1 min and showed a dose response identical to that of insulin receptor autophosphorylation. The PtdIns 3-kinase also associated with the insulin receptor in an insulin-stimulated manner, as approximately 50% of the total alpha PY-precipitable activity could be specifically immunoprecipitated with anti-insulin receptor antibody. Mutant insulin receptors displayed variable ability to stimulate the PtdIns 3-kinase, but in all cases the presence of PtdIns 3-kinase in alpha PY immunoprecipitates correlated closely with the tyrosyl phosphorylation of the endogenous substrate pp185. In CHO cells expressing a kinase-deficient mutant (IRA1018), there was no observable insulin stimulation of PtdIns 3-kinase activity in alpha PY immunoprecipitates and no tyrosyl phosphorylation of pp185. Substitution of Tyr1146 in the insulin receptor regulatory region with phenylalanine partially impaired receptor autophosphorylation, pp185 phosphorylation, and insulin-stimulated increases in alpha PY-precipitable

PtdIns 3-kinase activity. In contrast, a deletion mutant lacking 12 amino acids from the juxtamembrane region (IR delta 960) displayed normal in vivo autophosphorylation but failed to stimulate the PtdIns 3-kinase or phosphorylate pp185. Finally, a mutant receptor from which the C-terminal 43 amino acids had been deleted (IR delta CT) exhibited normal insulin-stimulated autophosphorylation, pp185 phosphorylation, and stimulation of the PtdIns 3-kinase activity in alpha PY immunoprecipitates. These data suggest that the PtdIns 3-kinase is itself a substrate of the insulin receptor kinase or associates preferentially with a substrate. A comparison of the biological activities of the mutant receptors with their activation of the PtdIns 3-kinase furthermore suggests that the PtdIns 3-kinase may be linked to insulin's ability to regulate DNA synthesis and cell growth.

L20 ANSWER 3 OF 3 MEDLINE on STN DUPLICATE 3

ACCESSION NUMBER: 92109680 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1722393
TITLE: Phosphorylation in vitro of the 85 kDa subunit of phosphatidylinositol 3-kinase and its possible activation by insulin receptor tyrosine kinase.
AUTHOR: Hayashi H; Miyake N; Kanai F; Shibasaki F; Takenawa T; Ebina Y
CORPORATE SOURCE: Department of Enzyme Genetics, University of Tokushima, Japan.
SOURCE: The Biochemical journal, (1991 Dec 15) Vol. 280 (Pt 3), pp. 769-75.
Journal code: 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 2 Mar 1992
Last Updated on STN: 3 Mar 2000
Entered Medline: 11 Feb 1992

AB Insulin causes a dramatic and rapid increase in phosphatidylinositol 3-kinase activity in the anti-phosphotyrosine immunoprecipitates of cells overexpressing the human insulin receptor. This enzyme may therefore be a mediator of insulin signal transduction [Endemann, Yonezawa & Roth (1990) J. Biol. Chemical 265, 396-400; Ruderman, Kapeller, White & Cantley (1990) Proc. Natl. Acad. Sci. U.S.A. 87, 1411-1415]. At least two questions remain to be elucidated. Firstly, does the insulin receptor tyrosine kinase phosphorylate phosphatidylinositol 3-kinase directly, or does it phosphorylate a protein associated with the 3-kinase? Second, if the enzyme is a direct substrate for the insulin receptor tyrosine kinase, does tyrosine phosphorylation of phosphatidylinositol 3-kinase by the kinase alter the specific enzyme activity, or does the amount of the tyrosine-phosphorylated form of the phosphatidylinositol 3-kinase increase, with no change in the specific activity? We report here evidence that the 85 kDa subunit of highly purified phosphatidylinositol 3-kinase is phosphorylated on the tyrosine residue by the activated normal insulin receptor in vitro, but not by a mutant insulin receptor which lacks tyrosine kinase activity. We found that an increase in enzyme activity was detected in response to insulin not only in the anti-phosphotyrosine immunoprecipitates of the cytosol, but also in the cytosolic fraction before immunoprecipitation. In addition, we partially separated the tyrosine-phosphorylated form from the unphosphorylated form of the enzyme, by using a f.p.l.c. Mono Q column. The insulin-stimulated phosphatidylinositol 3-kinase activity was mainly detected in the fraction containing almost all of the tyrosine-phosphorylated form.

This result suggests that tyrosine phosphorylation of phosphatidylinositol 3-kinase by the insulin receptor kinase may increase the specific activity of the former enzyme in vivo.

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=> d his

     (FILE 'HOME' ENTERED AT 14:06:24 ON 13 DEC 2006)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 14:07:00 ON 13 DEC 2006
L1            43 S "14171"
L2             4 S 14171/TI
L3             2 DUP REM L2 (2 DUPLICATES REMOVED)
L4       1527548 S KINASE?
L5             5 S L1 AND L4
L6             2 DUP REM L5 (3 DUPLICATES REMOVED)
L7        128031 S L4 AND (SERINE OR THREONINE)
L8       8068653 S CLON? OR EXPRESS? OR RECOMBINANT
L9         66403 S L7 AND L8
L10           75 S "T-P MOTIF"
L11           11 S L9 AND L10
L12            5 DUP REM L11 (6 DUPLICATES REMOVED)
L13      1018421 S (MODULAT? OR ACTIVAT? OR INHIBIT?)AND L4
L14       479153 S L8 AND L13
L15           24 S L10 AND L14
L16            7 DUP REM L15 (17 DUPLICATES REMOVED)
                 E KAPELLER-LIBERMANN
L17          114 S E2
L18            0 S L9 AND L17
L19           12 S L13 AND L17
L20            3 DUP REM L19 (9 DUPLICATES REMOVED)
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	L #	Hits	Search Text
1	L1	3	"14171" adj5 kinase$2
2	L2	77448	serine or threonine
3	L3	6293	l2 adj kinase$2
4	L4	914883	clon$3 or express$3 or recombinant
5	L5	1	"t-p motif"
6	L6	2453	l3 same l4
7	L7	977727	modulat$3 or bind$3
8	L8	2427	l6 and l7
9	L9	403	"14171"
10	L10	4	l8 and l9
11	L11	149	KAPELLER-LIBERMANN
12	L12	3	l9 and l11

	Issue Date	Pages	Document ID	Title
1	20040311	62	US 20040048305 A1	14171 Protein kinase, a novel human protein kinase and uses thereof
2	20030821	80	US 20030157082 A1	Methods and compositions for treating cancer using 140, 1470, 1686, 2089, 2427, 3702, 5891, 6428, 7181, 7660, 25641, 69583, 49863, 8897, 1682, 17667, 9235, 3703, 14171, 10359, 1660, 1450, 18894, 2088, 32427, 2160, 9252, 9389, 1642, 85269, 10297, 1584, 9525, 14124, 4469, 8990, 2100, 9288, 64698, 10480,20893, 33230,1586, 9943, 16334, 68862, 9011, 14031, 6178, 21225, 1420, 32236, 2099, 2150, 26583, 2784, 8941, 9811, 27444, 50566 or 66428 molecules
3	20031007	50	US 6630335 B1	14171 protein kinase, a novel human protein kinase and uses thereof

	Issue Date	Pages	Document ID	Title
1	20040311	62	US 20040048305 A1	14171 Protein kinase, a novel human protein kinase and uses thereof

	Issue Date	Pages	Document ID	Title
1	20051103	540	US 20050244834 A1	Single nucleotide polymorphisms in genes
2	20050609	215	US 20050125852 A1	Novel kinases
3	20040311	62	US 20040048305 A1	14171 Protein kinase, a novel human protein kinase and uses thereof
4	20030821	80	US 20030157082 A1	Methods and compositions for treating cancer using 140, 1470, 1686, 2089, 2427, 3702, 5891, 6428, 7181, 7660, 25641, 69583, 49863, 8897, 1682, 17667, 9235, 3703, 14171, 10359, 1660, 1450, 18894, 2088, 32427, 2160, 9252, 9389, 1642, 85269, 10297, 1584, 9525, 14124, 4469, 8990, 2100, 9288, 64698, 10480,20893, 33230,1586, 9943, 16334, 68862, 9011, 14031, 6178, 21225, 1420, 32236, 2099, 2150, 26583, 2784, 8941, 9811, 27444, 50566 or 66428 molecules

	Issue Date	Pages	Document ID	Title
1	20040311	62	US 20040048305 A1	14171 Protein kinase, a novel human protein kinase and uses thereof
2	20020117	75	US 20020006618 A1	Methods for using 20893, a human protein kinase
3	20031007	50	US 6630335 B1	14171 protein kinase, a novel human protein kinase and uses thereof